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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/802,955	03/16/2004	Eugene T. Michal	05618.P4124X	4196

8791 7590 05/17/2005

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EXAMINER	
FORD, ALLISON M	

ART UNIT	PAPER NUMBER
1651	

DATE MAILED: 05/17/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/802,955	MICHAL ET AL.	
	Examiner	Art Unit	
	Allison M. Ford	1651	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☐ Responsive to communication(s) filed on *06 April 2005*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-62* is/are pending in the application.

4a) Of the above claim(s) *9-18 and 23-62* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-8 and 19-22* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *16 March 2004* is/are: a)☐ accepted or b)☒ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

RD

DETAILED ACTION

Election/Restrictions

Applicant's election without traverse of Group I, claims 1-8 and 19-25, in the reply filed on 6 April 2005 is acknowledged. No election of species was included in the response; however, during a telephone conversation with Ms. Molleur on 13 April 2005, Ms. Molleur elected species I, claims 1-8 and 19-22 for initial examination, claims 23-25 thus become withdrawn as being directed to an unelected species. Claims 1-8 and 19-22 will be examined for patentability, with claims 1-62 remaining pending in the current application. Because the elections were made without traverse the restriction requirement is made FINAL.

Priority

Acknowledgement is made of applicant's claim for status as a CIP of copending application 10/414,602, filed 04/15/2003. However, no claim for priority under 35 USC § 120 has been made on the Oath/Declaration.

Drawings

The drawings are objected to as failing to comply with 37 CFR 1.84(p)(5) because the reference numbers in the specification do not match the reference numbers in the drawing (See, e.g. Brief Description of the Drawings Fig. 3A: lumen referred to as 300 in drawing, referred to as 301 in spec). Corrected drawing sheets in compliance with 37 CFR 1.121(d) are required in reply to the Office action to avoid abandonment of the application. Any amended replacement drawing sheet should include all of the figures appearing on the immediate prior version of the sheet, even if only one figure is being amended. Each drawing sheet submitted after the filing date of an application must be labeled in the top margin as either "Replacement Sheet" or "New Sheet" pursuant to 37 CFR 1.121(d). If the changes are not accepted

by the examiner, the applicant will be notified and informed of any required corrective action in the next Office action. The objection to the drawings will not be held in abeyance.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 2 and 3 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. Specifically, claim 2 recites genetically engineered cells in which at least one gene that encodes a polypeptide comprising an antigenic determinant that is recognized by a desired recipient subject has been disrupted, or wherein at least one gene that encodes a protein associated with the synthesis of a molecule comprising an antigenic determinant recognized by a desired recipient subject has been disrupted.

Regarding the cells in which at least one gene that encodes a polypeptide comprising an antigenic determinant that is recognized by a desired recipient subject has been disrupted, there is no evidence that any representative species of such a large and varied genera were in the possession of the inventors at the time of filing. To satisfy the written description aspect of 35 U.S.C. 112, first paragraph, for a claimed genus of molecules, it must be clear that: (1) the identifying characteristics of the claimed molecules have been disclosed, e.g., structure, physical and/or chemical characteristics, functional characteristics when coupled with a known or disclosed correlation between function and structure, or a combination of these; and (2) a representative number of species within the genus must be disclosed. The specification does not disclose any representative species of any cells in which a gene directly encoding a disclosed antigen

(which applicant calls an antigenic determinant that is recognized by a desired recipient subject), with or without identifying characteristics, such as nucleic acid or protein sequence information along with an identification of the specific epitope or antigen that is effected.

While applicants do disclose methods of scanning cDNA libraries to identify cells with such generic disrupted genes, such a method does not sufficiently describe the specific cell lines, or even the specific genes which are to be disrupted, much less the specific antigens or epitopes that are to be effected, which are required in order to claim such a broad genera. Scanning of a cDNA library is a common method well known in the art to identify almost any desired gene variants, but because it is so broadly applicable, it does not provide evidence that applicant had in their possession specific examples of the claimed invention. It is not described which specific cells applicant intends to use that satisfy the requirements of having at least one gene encoding a polypeptide comprising an antigenic determinant, which is recognized by a desired subject, being disrupted. Therefore, claims 2 and 3 (as claim 3 should be dependent on claim 2, see below) fail to satisfy the written description requirement.

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 2, 3, 8, and 19-22 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Applicant's claim 2 is directed to a method of identifying an infarct region within the ventricle of a subject; and delivering at least one structurally reinforcing component to the infarct region, wherein the structurally reinforcing component comprises genetically engineered cells, wherein at least one gene that encodes a polypeptide comprising an antigenic determinant that is recognized by a desired recipient

subject has been disrupted, or wherein at least one gene that encodes a protein associated with the synthesis of a molecule comprising an antigenic determinant recognized by a desired recipient subject has been disrupted.

As written the language of claim 2 is confusing, as it is not clear if the gene encoding a polypeptide comprising an antigenic determinant is to be disrupted, or merely included in the genetically engineered cell. It appears that applicant intends for either gene, upon inclusion in the genetically engineered cell, is to be disrupted; examination has been conducted as such.

Additionally, there is insufficient antecedent basis for the limitation "the desired recipient subject" in line 5 of claim 2. It would be remedial to refer to "a desired recipient subject."

Claim 3 recites the limitation "the at least one gene" in the first line of the claim. There is insufficient antecedent basis for this limitation in the claim. It appears claim 3 should be dependent on claim 2, not claim 1.

Applicant's claim 8 is directed to a method of identifying an infarct region within the ventricle of a subject; and delivering at least one structurally reinforcing component to the infarct region, wherein the structurally reinforcing component comprises cells, and further comprises at least one nucleic acid encoding a detectable polypeptide carried by the cells, the at least one nucleic acid being operably linked to a promoter. It is not clear what is to be done with the nucleic acid encoding the detectable polypeptide; claim 1 is directed to a method, claim 8 does not provide an additional method step, nor does it limit any of the steps recited in claim 1. It appears the claim intends for the cells of claim 1 to further comprise at least one nucleic acid encoding a detectable polypeptide carried by the cells, wherein the at least one nucleic acid is operably linked to a promoter; examination has been carried out as such.

Applicant's claim 19 is directed to a method comprising identifying an infarct region within the ventricle of a subject; applying a pacing algorithm for CRT (cardiac resynchronization therapy) treatment, or normal pacing; and delivering at least one structurally reinforcing component to the infarct region. As

written the language of claim 19 is confusing. It is not clear if the second step is to comprise applying a pacing algorithm for either a) CRT treatment or b) for normal pacing; or if normal pacing is not connected to the pacing algorithm, but is accomplished by another means; finally, it is not clear if normal pacing is a treatment at all, or if it is the result of the CRT treatment. Claims 20-22 are dependent on claim 19 and thus are rejected on the same basis.

Applicant's claim 21 is directed to the method of claim 20, wherein the structurally reinforcing component comprises genetically engineered cells, wherein at least one gene that encodes a polypeptide comprising an antigenic determinant that is recognized by a desired recipient subject has been disrupted, or wherein at least one gene that encodes a protein associated with the synthesis of a molecule comprising an antigenic determinant recognized by a desired recipient subject has been disrupted.

As in claim 2, the language of claim 21 is confusing, as it is not clear if the gene encoding a polypeptide comprising an antigenic determinant is to be disrupted, or merely included in the genetically engineered cell. It appears that applicant intends for either gene, upon inclusion in the genetically engineered cell, is to be disrupted; examination has been conducted as such.

Additionally, there is insufficient antecedent basis for the limitation "the desired recipient subject" in line 5 of claim 21. It would be remedial to refer to "a desired recipient subject." Claim 22 is dependent on claim 21 and is thus rejected on the same basis.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 1, 2, 5, 6, 7, and 8 are rejected under 35 U.S.C. 102(b) as being anticipated by Etzion et al (J Mol Cell Cardiol, 2001), in light of Lewin (Genes VII, 2000).

Etzion et al teach inducing myocardial infarction in the ventricle of rat subjects; identifying the infarct region visually on the basis of a surface scar and wall motion akinesis; and delivering embryonic rat cardiomyocytes to the infarct region (See Pg. 1322) (Claim 1). Cell transplantations were performed seven days after myocardial infarction (See Pg. 1322, col. 2) (Claim 7). Representative samples of the transplanted cells were transfected with recombinant adenovirus carrying the nuclear reporter gene *LacZ*, encoding for β-galactosidase, X-gal staining revealed blue nuclei, indicating that the *LacZ* gene was expressed; the *LacZ* must be operably linked to an operator for proper expression; therefore the nucleic acid encoding the detectable β-galactosidase was operably linked to a promoter (See Pg. 1324, col. 2 and Fig. 1(d) & Lewin Pg. 277-280) (Claim 8).

Though Etzion et al teach the transplanted myocardiocytes engraft into and are able to survive in the infarcted myocardium and increase wall thickness and reduce wall stress, they do not specifically state the engrafted cells replace damaged cells in and around the infarct region or that the cells increase the modulus of elasticity of the infarct region. However, because Etzion et al teach the same process of delivering cells to an infarct region of a subject, as claimed in the current application, the resulting infarct region is one and the same as in the current application, and thus the engrafted cells replace the damaged cells in and around the infarct region and increase the modulus of elasticity of the infarct region the same as in the current application (Claims 5 and 6). Therefore the reference anticipates the claimed subject matter.

Claims 1, 5, 6 and 7 are rejected under 35 U.S.C. 102(b) as being anticipated by Leor et al (Cardiovascular Research, 1997).

Leor et al teach identifying an infarct region within the ventricle of rat subjects and delivering cultured human fetal ventricle cells or fetal rat ventricle cells into the scar tissue of infarct region (which applicant calls delivering at least one structurally reinforcing component to the infarct region) (Claim 1). The human fetal ventricle tissue (comprising human fetal ventricle cells) was engrafted as early as 7 days after infarction; the fetal rat ventricle tissue (comprising fetal rat ventricle cells) was engrafted as early as 9 days after infarction (See Pg. 437) (Claim 7). Additionally, though Leor et al does not specifically state the engrafted ventricle cells replace damaged cells in and around the infarct region or that the cells increase the modulus of elasticity of the infarct region, Leor et al teach the same process of delivering cells to an infarct region of a subject, as claimed in the current application, and thus the resulting infarct region is one and the same as in the current application, therefore the engrafted ventricle cells replaced the damaged cells in and around the infarct region and increase the modulus of elasticity of the infarct region just as in the current application (Claims 5-6). Therefore the reference anticipates the claimed subject matter.

Claims 1, 5, 6 and 7 are rejected under 35 U.S.C. 102(b) as being anticipated by Pouzet et al (Circulation, 2001).

Pouzet et al teach creating, and therefore identifying, an infarct region within the left ventricle of rat subjects and delivering skeletal muscle cells to the infarct region (which applicant calls delivering at least one structurally reinforcing component to the infarct region) (Claim 1). The skeletal muscle cells were delivered one week after infarction (Claim 7). Additionally, though Pouzet et al does not specifically state the engrafted ventricle cells replace damaged cells in and around the infarct region or that the cells increase the modulus of elasticity of the infarct region, Leor et al teach the same process of delivering cells to an infarct region of a subject, as claimed in the current application, and thus the resulting infarct region is one and the same as in the current application, therefore the engrafted ventricle

cells replaced the damaged cells in and around the infarct region and increase the modulus of elasticity of the infarct region just as in the current application ((Claims 5-6) (See Pg. I-224). Therefore the reference anticipates the claimed subject matter.

Claims 1, 5, and 6 are rejected under 35 U.S.C. 102(b) as being anticipated by Soykan et al (US Patent 6,151,525).

Soykan et al teach a method comprising identifying an infarct region within the ventricle of a subject, and performing cellular cardiomyoplasty, which involves delivering cells to the infarct region, in conjunction with electrical stimulation (See col. 4, ln 33-44) (Claim 1). The method restores elasticity and contractility to the tissue (See col. 4, ln 59-65). Additionally, though Soykan et al teach contractility and elasticity are restored to the tissue, they do not specifically state the engrafted cells replace damaged cells in and around the infarct region or that the cells increase the modulus of elasticity of the infarct region; however, because Soykan et al teach the same process of delivering cells to an infarct region of a subject, as claimed in the current application, and thus the resulting infarct region is one and the same as in the current application, the engrafted cells replaced the damaged cells in and around the infarct region and increase the modulus of elasticity of the infarct region just as in the current application (Claims 5-6). Therefore the reference anticipates the claimed subject matter.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 2-4 are rejected under 35 U.S.C. 103(a) as being unpatentable over Etzion et al (J Mol Cell Cardiol, 2001), in view of Gustafsson et al (US Patent 6,153,428).

Etzion et al teach inducing myocardial infarction in the ventricle of rat subjects; identifying the infarct region visually on the basis of a surface scar and wall motion akinesis; and delivering embryonic cardiomyocytes to the infarct region (See Pg. 1322).

Etzion et al teach performing allogeneic transplants from embryonic rats to adult rats; however, even in their syngeneic transplantations immune reactions caused rejection problems (See Pg. 1329, col. 1).

Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to use cells or cell lines that are immunogenically tolerable to the recipient, so as to not elicit an immune reaction.

Gustafsson et al teach such a cell line, transgenic α-1,3-galactosyltransferase (GGTA1) knock-out swine cells, that is immunogenically tolerable to recipients, even in xenotransplantations (See Gustafsson et al col. 2, ln 19-33). In the transgenic α-1,3-galactosyltransferase (GGTA1) knock-out swine cells the genes, on one or both alleles, has been disrupted to reduce the amount of α-1,3-galactosyltransferase produced to an extent sufficient to prevent the cells' ability to provide carbohydrates with the Galα1-3Galβ1-4GlcNAc epitope from being provided to the cell surface, thereby rendering the cells immunogenically tolerable to the intended recipient. Whole animals or individual cells and/or tissues of any desired type can be produced in the GGTA1 knock-out phenotype, with one or both alleles having been disrupted. Gustafsson et al teach the transgenic GGTA1 knock-out cells can be used as a source of cells for transplantation (See Gustafsson et al, col. 5, ln 58-col. 6, ln 9).

Therefore, one of ordinary skill in the art, at the time the invention was made, would have been motivated to use GGTA1 knock-out myocardiocyte cells, taught by Gustafsson et al, in the method of Etzion et al, in order to prevent immune rejection (Claims 2 and 4). One of ordinary skill in the art would

have been especially motivated to use the GGTA1 knock-out myocardiocytes of Gustafsson et al in the method of Etzion et al, if the cells were being delivered to a human subject as treatment of a myocardial infarction because Gustafsson et al teach the Galα1-3Galβ1-4GlcNAc epitope is the major target for anti-swine xenoreactive human natural antibodies (See Gustafsson et al, col. 1, ln 38-44). One would have expected success using swine GGTA1 knock-out myocardiocytes, developed by Gustafsson et al, in the method of Etzion et al because Etzion et al teach successfully transplanting the embryonic myocardiocytes into the infarct region in the ventricle of a subject; however Etzion et al did report problems associated with immune reaction from the allogeneic cells. Using the immunogenically tolerable GGTA1 knock-out swine cells of Gustafsson et al, one would expect elimination of the immune reaction, and overall success of the transplant.

Gustafsson et al teach that heterozygous or homozygous GGTA1 knock-out swine can be produced (homozygous meaning both chromosomal copies of the α-1,3-galactosyltransferase gene are disrupted) (See col. 6, ln 2-5). Therefore, it would have been obvious to one of ordinary skill in the art to use a cell line derived from a homozygous GGTA1 knock-out swine, wherein both chromosomal copies of the GGTA1 gene are disrupted, in order to ensure that α-1,3-galactosyltransferase is not produced in amounts sufficient to produce the Galα1-3Galβ1-4GlcNAc epitope (Claim 3). By disrupting both chromosomal copies there is no chance one copy could compensate and produce a sufficient amount of α-1,3-galactosyltransferase. One of ordinary skill in the art would have been motivated to use a cell line wherein both chromosomal copies of the GGTA1 gene have been disrupted in order to ensure GGTA1 is not produced in significant amounts. One would have expected success because Gustafsson et al teach that transgenic animals and cells can be produced where either one or both copies of the alleles are disrupted (See Gustafsson et al, col. 5, ln 58-col. 6, ln 9).

Therefore the invention as a whole would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made.

Claims 19-22 are rejected under 35 U.S.C. 103(a) as being unpatentable over Soykan et al (US Patent 6,151,525), in view of Chachques (US 2002/0124855 A1), further in view of Gustafsson et al (US Patent 6,153,428).

Soykan et al teach a method of reversing the damage to necrotic heart muscle following myocardial infarction, comprising identifying an infarct region within the ventricle of a subject, and performing cellular cardiomyoplasty, which involves delivering cells to the infarct region, in conjunction with electrical stimulation (See col. 4, ln 33-44). The method restores elasticity and contractility to the tissue (See col. 4, ln 59-65). Soykan et al teach the electrical stimulation device is to provide electrical pulses at the correct time to make the new tissue beat in synchrony with the rest of the heart muscle, and can include conventional implantable pulse generators or burst generator circuits (See col. 13, ln 10-col. 15, ln 63).

Though Soykan et al do not specifically teach applying a pacing algorithm for cardiac resynchronization therapy treatment or normal pacing, Chachques et al teach a similar method of applying electrical stimulation to newly implanted cells in an area of infarction using pacing algorithms for cardiac resynchronization therapy (See Chachques et al, Pg. 2-3, paragraphs 0028-0032) (Claims 19 and 20). Chachques et al teach the cardiac resynchronization therapy, involving the pacing algorithms, corrects asynchronous beating of the lower chambers of the heart, often caused by the weakened muscles due to the infarction. Therefore, one of ordinary skill in the art at the time the invention was made would have been motivated to use the electrical stimulation device of Soykan et al to apply a pacing algorithm for cardiac resynchronization therapy, or normal pacing, as taught by Chachques et al. One of ordinary skill in the art would have been motivated to apply the pacing algorithms described by Chachques et al in the method of Soykan et al in order to synchronize the beating of the newly implanted cells as well as the chambers of the damaged heart, in order to induce proper heart beating. One would have expected

success applying a pacing algorithm using the electrical stimulation device of Soykan et al because Soykan et al teach any suitable electrostimulation devices can be used to properly stimulate the heart muscle contractions.

Finally, though Soykan et al do not teach using genetically engineered cells in which at least one gene that encodes a polypeptide comprising an antigenic determinant that is recognized by a desired recipient subject has been disrupted, or a genetically engineered cell in which at least one gene that encodes a protein associated with the synthesis of a molecule comprising an antigenic determinant recognized by a desired recipient subject has been disrupted, it would have been obvious to one of ordinary skill in the art at the time the invention was made to use such immunogenically tolerable cells when performing non-syngeneic transplantations, for example, xenotransplantations, in order to reduce immune reactions.

Gustafsson et al teach a cell line, transgenic α-1,3-galactosyltransferase (GGTA1) knock-out swine cells, that is immunogenically tolerable to recipients, especially in xenotransplantations wherein humans are the recipients (See Gustafsson et al col. 2, ln 19-33). See teachings above. Therefore one of ordinary skill in the art would have been motivated to use the GGTA1 knock-out swine cell line of Gustafsson et al in the method of Soykan et al in view of Chachques et al, which comprises identification of an infarct region within the ventricle, delivery of cells to the area of infarct, and application of a pacing algorithm for CRT, in order to prevent immune rejection of the transplanted cells in cases of xenotransplantation, especially wherein humans are the recipients (Claims 21 and 22). One would have expected success because Soykan et al teach cells can be genetically manipulated before transplantation (See col. 7, ln 60-63) and Gustafsson et al the genetically engineered cells can be used for transplantation (See Gustafsson et al col. 5, ln 58-col. 6, ln 9), and Soykan et al teach that the combination of cellular cardiomyoplasty and electrical stimulation results in increased restoration of function in the myocardium, and decreased

morbidity and mortality (See col. 4, ln 33-65). Therefore the invention as a whole would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made.

Double Patenting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. See *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and, *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent is shown to be commonly owned with this application. See 37 CFR 1.130(b).

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Claims 1, 5 and 7 are provisionally rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1, 2, and 4 of copending Application No. 10/414,602. Although the claims of copending Application 10/414,602 are not identical, they are not patentably distinct from the current application because it would have been obvious to one of ordinary skill in the art to deliver an implant comprising cells to the infarct region for the purpose of reinforcing the weakened infarct region. One of ordinary skill in the art would have been motivated to deliver an implant comprising cells in order to rebuild and strengthen the weakened area, the cells provided in the implant would then be expected to develop into new tissue to rebuild the region.

Additionally, claims 1 and 5 are provisionally rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 2 and 3 of copending Application No. 10/414,767. Although the claims of copending Application 10/414,767 are not identical, they are not patentably distinct from the current application because it would have been obvious to one of ordinary skill in the art to deliver an implant comprising cells as the solid material to the infarct region for the

purpose of reinforcing the weakened infarct region and increasing the compliance of the ventricle. One of ordinary skill in the art would have been motivated to deliver an implant comprising cells in order to rebuild and strengthen the weakened area, the cells provided in the implant would then be expected to develop into new tissue to rebuild the region.

These are provisional obviousness-type double patenting rejections because the conflicting claims have not in fact been patented.

Conclusion

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Allison M. Ford whose telephone number is 571-272-2936. The examiner can normally be reached on 7:30-5 M-Th, alternate Fridays.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Michael Wityshyn can be reached on 571-272-0926. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Allison M Ford
Examiner
Art Unit 1651

LEON B. LANKFORD, JR.
PRIMARY EXAMINER